architecture of the kidney cortex was displaced by the invasive neoplastic tissue. All other organs were within normal limits. Because of the primary site of the neoplasm, the formation of acini, the extensive fibrosis, and the metastasis by arterioles and lymphatics, the mass was considered to be a mammary adenocarcinoma with metastasis to lymph nodes, muscle, lung, kidney, and parietal pleural surface.

The Père David’s deer is extinct in its Chinese homeland and has survived only in captivity, which has been a source of stock for release into wildlife preserves. Because of the endangered status of this cervid, it is important to document cases of mortality in this species as part of a data base for managing the Père David’s deer.

This is the first report of a metastatic mammary adenocarcinoma in the Père David’s deer. Most reports of neoplasia in wild deer are of fibropapillomas or squamous cell carcinomas, however, there was 1 report of a fibrosarcoma in a Père David’s deer. Mammary neoplasias have not been reported in wild deer, and they are rare in other ruminants. In this case, the neoplasia behaved aggressively as seen in mammary neoplasias in other species. Not only did it destroy all normal architecture in the area of the mammary gland, but it invaded lymph nodes, muscle, lung, and kidney.

References

Hereditary hypermetria in Shorthorn cattle

Ana L. Schild, Franklin Riet-Correa, Maria C. Mkndez, Severo S. Barros

A neurologic disease characterized by permanent bilateral symmetric hypermetria was observed in a Shorthorn herd in the state of Rio Grande do Sul, southern Brazil. During gait, the ataxic animals showed marked extension of the limbs, especially the forelimbs. When the animals were forced to run, a more pronounced hypermetria with loss of balance was observed (Fig. 1). Falling and tremors of the head and neck occasionally occurred in the more severely affected calves. The disorder was not progressive, and calves were affected from birth. All affected calves were able to suck, and the disease did not affect their normal development. Death could eventually occur due to misadventure.

The disease apparently appeared in 1980, after the utilization on the farm of a bull with discrete hypermetria. From 1980 to 1989, about 15 calves showed signs of the disease out of approximately 2,000 calves born during this period. The bulls used in the herd were produced on the same farm in which the disease occurred.

The epidemiologic data suggested an inherited etiology for the disease. To confirm this possibility, a test mating was performed. One bull (I-1) with signs of the disease was mated to 16 cows from a Shorthorn herd where the disease had not been observed and with 11 cows from other breeds, including Charolais, Holstein, and crossbreeds (I-2-28). Fifty-seven calves (11-1-57) without symptoms of the disease were born from this mating. The same bull was later mated to 24 of his daughters. Out of 34 calves born (111-1-34), 17 (8 males and 9 females) were affected by congenital hypermetria (Fig. 2).

Figure 1. Calf with signs of hypermetria.

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Six affected calves were killed by exsanguination and necropsied. Three of them, ages 20 days, 45 days, and 9 months, were born at the farm; the other 3, ages 1, 3, and 3 months, resulted from the test mating. Macroscopically, no abnormalities were detected in the nervous system and other organs. Cerebellum weights, total brain weights, and their ratios obtained in 3 calves are presented in Table 1. In the other calves, the brain was not weighed because immediately after death, samples of the cerebellum were fixed for electron microscopic examination. For histologic study, fragments of all organs and nervous system, including cerebral cortex, internal capsule, basal ganglia, quadrigeminal tubercles, pons, medulla oblongata, spinal cord, and cerebellum (caudal vermis, uvula, nodulus, lingula, rostral vermis) and cerebellar peduncles, were fixed in buffered neutral 10% formalin, embedded in paraffin, sectioned at 6 µm, and stained with hematoxylin and eosin and luxol fast blue. For electron microscopic evaluation, fragments of cerebellum, including cortex and regions of fastigial, interposital, and lateral cerebellar nucleus, from 3 calves were fixed in cacodylate-buffered glutaraldehyde, postfixed in osmium tetroxide, dehydrated in upgraded alcohols, and embedded in Epon. Semithin sections were stained with methylene blue, and ultrathin sections were doubly stained with lead citrate and uranyl acetate.

No significant histologic lesions were observed in the nervous system or other organs, and no significant ultrastructural changes were observed in cerebellar cortex or cerebellar nucleus.

The clinical signs observed indicate a cerebellar syndrome. The history and genetic analysis indicate that the disease is due to an autosomal recessive gene. This conclusion is based in the 17 (50%) affected calves that resulted from matings of the affected homozygous bull with his heterozygous daughters (Fig. 2). This number is identical to the number expected when an autosomal recessive gene is involved.

Cerebellar abiotrophy is another cerebellar syndrome characterized by degeneration of Purkinje cells. The lesion is due to an intrinsic abnormality in the metabolic structure of these neurons, which preclude their survival. The disease has been frequently described in cattle and other species. The clinical signs appear at different times after birth and are rapid or slowly progressive. Probable cerebellar abiotrophy and hypoplasia also have been reported in Hereford cattle with nonprogressive ataxia present at birth. In a similar disease known as bovine familial convulsions and ataxia in

<table>
<thead>
<tr>
<th>Calf no.</th>
<th>Age</th>
<th>Cerebellum weight (g)</th>
<th>Brain weight (g)</th>
<th>Ratio</th>
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<tr>
<td>1</td>
<td>9 months</td>
<td>33.50</td>
<td>346.20</td>
<td>0.0967</td>
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<tr>
<td>2</td>
<td>1 month</td>
<td>27.54</td>
<td>281.10</td>
<td>0.0979</td>
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<tr>
<td>3</td>
<td>20 days</td>
<td>23.15</td>
<td>251.18</td>
<td>0.0921</td>
</tr>
</tbody>
</table>

* Normal cerebellum/brain weight ratio is 0.083–0.11.
Ubiquitin immunocytochemistry of spinal cord in an inherited porcine motor neuron disease

Donal O’Toole, Val Welch, Kari Redland

Ubiquitin is a 76-amino acid polypeptide that is highly conserved in all eukaryotic cells. It is a member of the heat-shock (cell stress) family of proteins, and it shows high levels of expression when cells are subject to specific stresses such as heat, hypoxia, irradiation, and viral infection. A major function of ubiquitin involves covalent attachment to target proteins (“ubiquitination”), thereby designating ubiquitin-conjugated proteins for ATP-dependent nonlysosomal proteolysis. Some ubiquitinated proteins may undergo cataplasmatosis via the lysosomal system. Immunocytochemical studies using antibodies to ubiquitin have documented ubiquitinated inclusions in selected human neurodegenerations, as well as in some chronic hepatic and muscular disorders. Ubiquitin-positive intraneuronal inclusions are now recognized in Alzheimer’s disease, Down’s syndrome, diffuse Lewy body disease, amyotrophic lateral sclerosis (ALS), infantile motor neuron disease, and normal aged brains, among others. It is unclear whether ubiquitin is an integral part of these inclusions or if instead it is tagging proteinaceous inclusions to attempt catabolism. Detection of the intracytoplasmic skein-like or granular ubiquitin-positive inclusions, some of which are closely associated with Bunina bodies, in spinal cord motor neurons from human ALS patients has been advocated as the most reliable way to confirm histologically a clinical diagnosis of ALS.

There are few studies of ubiquitin expression in the neuraxis of animals with chronic neurodegenerations. Hereditary porcine neuronal system degeneration (HPNSD) is a genetically determined lower motor neuron disease of pigs that was originally identified in England. Typical clinical signs involve paraparesis of the pelvic limbs, with quadruparesis in some severely affected pigs. Alpha motor neurons in the spinal cord become atrophic, and many develop intracytoplasmic vacuoles before they disappear (Fig. 1). This is a rare, dominantly inherited trait that has been recognized only in pigs in England and in a colony of pigs kept initially at the Central Veterinary Laboratory in Weybridge, England (1983-1991) and then at the Wyoming State Veterinary Laboratory (WSVL) (1992-the present). The subtlety of histologic lesions in the spinal cord may hamper diagnosis. The purpose of this study was to determine, using an immunohistochemical method and 5 antibodies to conjugated ubiquitin, whether abnormal ubiquitin patterns occurred in the spinal cords of 4 piglets with typical clinical signs of HPNSD. Documentation of such inclusions could be useful diagnos-

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